PHYSIOLOGICAL SPECIALIZATION OF *MELAMPSORA LINI* (PERS.) LÉV. IN AUSTRALIA.

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(Plates iii–iv; three Text-figures.)

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Synopsis.

A physiologic race survey of the pathogen *Melampsora lini* (Pers.) Lév. was carried out at Sydney University over the period 1948 to 1953. Several important new races were recorded indicating a marked change in the pathogenicity of the pathogen in Australia since the first surveys undertaken by Waterhouse and Watson. Particular attention is drawn to the difference in host range and geographic distribution of the Punjab-attacking and non-Punjab-attacking race groups.

Introduction.

Physiologic specialization in *M. lini* was first demonstrated by Flor in 1935, although others were aware of physiologic differences within the species as early as 1865. By 1935 Flor had identified fourteen races on nine differential varieties. Since then Flor has modified and increased his differential series on which he had differentiated 127 races by 1945 (Flor, 1945). Other races were detected by workers in Canada, Europe, Australia, South America and India (Straib, 1939; Waterhouse and Watson, 1943; Vallega, 1944; Prasada, 1948). The differential varieties currently used by Flor are monofactorially resistant or immune to the races identified by him, and could effectively differentiate over a million races which might be realized by random recombination of genes already detected in the pathogen.

According to McAlpine (1906), *M. lini* was first identified in Australia in 1889 on cultivated flax *Linum usitatissimum*. It was also common on native flax *L. marginale*. He assumed that it had been introduced from overseas.

Flax and linseed, especially flax, have been grown on a small scale since the earliest colonial days. Acreages remained very small until the 1939-1945 war. Prior to 1939 the yearly sowings of flax averaged less than 2,000 acres. This increased to a peak of 61,000 acres in 1944, followed by a progressive decline to acreages of less than 5,000 acres between 1950 and 1952. Commercial crops of flax were limited to Victoria, South Australia, Western Australia and Tasmania.

Blue Riga, the main flax variety in Victoria prior to 1937, and Concurrent and Liral Crown, the dominant varieties in the southern States during and after the war years, were highly susceptible to the majority of the Australian races identified since 1943. Rust-resistant varieties began to replace them (Thomas and Millington, 1946), but the pathogen continued to be a serious hazard to flax crops (Cass Smith and Harvey, 1946).

Linseed acreages also increased during the war years and remained at a moderately high level for some time afterwards. Moderate acreages were grown in New South Wales and Queensland as well as the southern States as late as 1953, when these studies were closed. The first commercial crops of Punjab suffered severely from rust, but the linseed growing industry was reestablished with Walsh, which has been generally resistant to *M. lini* in all States since its introduction.

Following serious outbreaks of rust in the early war years Waterhouse and Watson (1941 and 1943) commenced a survey of the pathogen. Flor's series of eleven differential varieties and Argentine 705-1 constituted the first set of differential varieties. The first survey keyed out a unique race which differed significantly from

all overseas races in its avirulence on Bison, a variety susceptible to all these races. It was designated race A, and was highly virulent on Punjab. Later studies determined five additional races, B to F. Most of the collections of rust were from commercial crops, but several were from *L. marginale*. The latter, with one exception, were identified as races A or F, both Punjab-attacking and avirulent on Bison. Watson continued the survey beyond 1943 but failed to detect any significant change in the rust population up to 1945. A further survey was carried out by Charles in 1947 on an extended host series of eighteen varieties including the original twelve. This was continued by Kerr in 1948 prior to the project dealt with in this paper. These studies determined seven additional races G to O. In addition, Charles reclassified the original six races A to F, adding to their description reactions given by them on six additional varieties (Millikan, 1951).

Experimental Methods and Material.

The differential series previously used by Charles (1947) was adopted during the current studies. To these were added Koto, a variety showing promise as a useful immune parent, and a selection of Walsh, the only commercial variety of linseed grown in Australia. Owing to impurity Williston Brown was later dropped from the series. The differential varieties are listed below.

Variety.	Sydney University Accession Number.	C.I. Number.
Buda	Fx 1	—
Williston Golden	Fx 2	25–1
Akmolinsk	Fx 3	515–1
J.W.S.	Fx 4	708–1
Abyssinian	Fx 5	701
Kenya	Fx 6	709–1
Argentine	Fx 7	705–1
Very Pale Blue Crimped	Fx 9	647–1
Ottawa 770B	Fx 10	355
Argentine	Fx 11	462
Bison	Fx 13	389
Punjab	Fx 14	—
Walsh	Fx 86	—
Morye	Fx 318	112
Newland	Fx 319	188
Bolley Golden	Fx 320	644
Italia Roma	Fx 321	1005–1
Leona	Fx 322	836
Tammes' Pale Blue	Fx 323	333–1
Koto	Fx 326	842

Collections of rust were received from Queensland, New South Wales, Victoria, South Australia and Western Australia. The collections were received during spring and early to mid-summer. They were cultured on the unnamed variety F257, which was found to be more susceptible to the Australian collections than the previously used variety, Concurrent. The uredospore inoculum was collected and stored in small, glass, cork-stoppered phials in a refrigerator at 0° to 2°C.

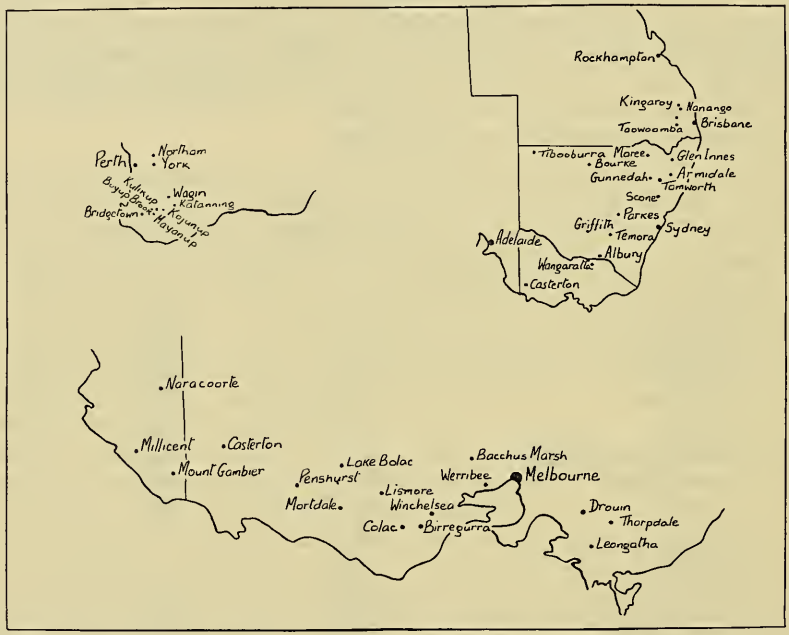
The studies were carried out in the glasshouses at Sydney University during the cooler months of the year from April to late October. Temperature and light intensity and duration fluctuated according to conditions out of doors. This was offset by the occasional use of radiators and incandescent lights during winter, when the equipment was available.

The reaction of the differential varieties was read after about 10 to 14 days, depending on conditions during incubation. Flor's system of reaction classification was adopted as far as possible, but the wide range of reaction induced by fluctuations

in the environment prevented very fine distinctions. Only three types of reaction were finally distinguished: immune (including consistently immune or highly resistant), resistant (including the wide range of rather variable intermediate reactions from resistant to moderately susceptible), susceptible (including highly susceptible reactions sometimes depressed to moderate susceptibility by adverse environmental conditions). (See Plate iii.)

Each collection was tested at least twice, and particular attention was paid to the varieties giving reactions rather sensitive to the environment. The varietal screening method was used to confirm the occurrence of new races, along with the use of single spore cultures.

Adequate precautions were taken to prevent contamination of the races in the glasshouse. Races were cultured where possible on varieties immune to the other races



Text-fig. 1.—Maps of linseed and flax growing States indicating main flax and linseed growing districts, and districts from which *Melampsora lini* specimens were received.

cultured in the same house. They were subcultured no more than twice a year and sometimes only once a year.

The spore dusting inoculation technique used by Flor (1935) and Waterhouse and Watson (1941) did not always give satisfactory results during these studies. A more laborious method was adopted, but this resulted in consistently heavy infection. A drop of water was placed on the crown of young unfolded leaves of each seedling. Uredospores were spread on the surface of water in a petri dish and transferred by spatula to the seedling tip. At first poor germination of uredospores stored for more than two months sometimes necessitated reinoculation. It had been the practice to check the percentage germination of the residual spores left in the petri dish. While germination was generally good on the seedling tips, it was not uncommon to discover that the spores had generally failed to germinate on the water in the petri dish. This was attributed to diffusion of substances from the host tissue into the terminal drop of water. Boiled aqueous host extract was found to induce consistently maximum germination in stored spores sown on the surface of the extract. Experiment showed that it was generally sufficient to spread stored spores on the surface of concentrated

host extract before transfer to the terminal drop of water on the seedling to ensure good germination. When spores had been stored for long periods it was sometimes sufficient to leave the spores on concentrated host extract for several minutes before transfer to the seedling. To ensure good germination host extract was used to moisten the seedling tips.

The excised shoot technique was used during most of these studies to conserve bench space and ensure a ready supply of seedling tips for inoculation (Kerr, 1951).

During these studies a technique was developed to preserve rust reactions for comparison with results obtained later. Leaves were preserved under Scotch tape on semi-absorbent paper and stored in the dark in a refrigerator at 0° to 2°C. After two years there was no appreciable deterioration, the leaves and reaction closely resembling freshly collected material.

Experimental Results.

The Effect of the Condition of the Shoot on the Rust Reaction.

The condition of the host during the incubation period played an important role in determining the reaction of most varieties. The most susceptible varieties usually developed a measure of resistance as the shoots became more fibery with age.

Bison was fully susceptible to every Australian race, except race 1, and F257 was fully susceptible to every race in the early seedling stage. Race 6 was cultured on both varieties under identical conditions, on young succulent excised shoots of F257 which remained succulent during the incubation period, and on older excised shoots of Bison which were quite succulent when inoculated, but became rather fibery later. Both varieties gave a susceptible reaction after 10 days. After three weeks the infected Bison leaves had withered and yielded very little inoculum. F257 continued to yield very heavy loads of uredospores. A quantitative assessment of the degree of active infection at this time was given by the number of excised stems of each variety with prominent uredosori. Only fourteen of thirty-two excised shoots of Bison had uredosori on the stem. Thirty-nine of the forty-one shoots of F257 showed prominent uredosori on the stems. Bison had obviously acquired some measure of resistance to race 6.

The degree of succulence of the inoculated shoot was more important than the age of the parent plant in determining reaction. Since succulence was largely determined by environmental factors, rather than any inherent characteristic of the host, during the first month or so after excision of the shoot, the value of standardizing growing conditions at the optimal was apparent.

The Effect of the Environment on Host Reaction.

Recognition of the significantly different reactions of each variety in the differential series to different races of rust is fundamental to the success of any physiologic race survey. Since no two varieties behave alike, each must be studied individually to determine the number of significantly different reactions and the extent to which each reaction is subject to environmentally induced fluctuations.

The lack of temperature and light control facilities at Sydney accentuated the importance of this aspect of the work. Variations in the environment often induced changes in reaction as great as that considered adequate for the differentiation of races. The reaction of several varieties to both races 2 and 13 was suppressed in the direction of greater resistance under the very warm incubation conditions in the glasshouse during late October and early November. The variety Very Pale Blue Crimped was immune to race 2 under these conditions, although it gave a fully susceptible reaction to race 2 under the optimal conditions (15° to 20°C.) prevalent earlier in the year.

Races 1 and 2 were tested in the field at Sydney to determine their capacity to survive in the uredospore stage during spring and summer. The former was highly virulent on Punjab and moderately virulent on F257 in the glasshouse under optimal conditions, and the latter was avirulent on Punjab but highly virulent on F257. Both were well established on their susceptible hosts in September. Periodic sowings of

both varieties were made to ensure a constant supply of young infectable plants. As the temperature increased during November and December, Punjab continued to be heavily infected by race 1. The poor infection of F257 reflected the lack of tolerance of race 2 to high summer temperatures and the relative immunity of the variety to race 1 under these conditions.

Reaction Levels.

After careful consideration of all the results obtained with each accession it was decided to recognize no more than three distinctly different reactions in each variety. In most cases only two significantly different types of reaction were differentiated. Some varieties may have had more significantly different reactions than the number finally attributed to them. But in view of the wide variations in light intensity and temperature during tests and between different tests of each accession, it was considered unwise to postulate a greater number than that finally decided upon.

It is proposed to designate significantly different reactions within a variety "reaction levels". There seems to be no conclusive evidence to show whether the reaction of a variety to all possible races of a given pathogen ranges by almost imperceptible stages from complete immunity to complete susceptibility, or whether there is only a limited number of different reactions. If the former is the case the term reaction level has little meaning, the number of levels determinable being limited only by the degree of environmental control and accuracy of observation. The results obtained during this and most other rust surveys, and Flor's genetic investigations of virulence and avirulence in *M. lini* seem, however, to support the thesis that there is only a limited number of reaction levels. This assumption underlies the use of the term reaction levels.

In the final analysis races should be differentiated from each other and described in terms of these reaction levels. A double descriptive system would seem to be necessary. The rust reaction of a variety to any race is the product of the interaction of a gene or genes in the pathogen and corresponding genes in the host, modified by the environment, and possibly also by modifying genes in the race and host. No two varieties are likely to have the same range of reactions. The resistant reaction of Walsh was characteristic of that variety and differed noticeably from the resistant reaction of Akmolinsk to the Australian races. The initial descriptive terminology should be sufficiently comprehensive to define these differences.

However, fine differences in the resistant reaction of varieties are irrelevant in the final race classification. These differences are no more relevant than those induced by the environment. Final decisions on race status must necessarily be intravarietal. Two races can be differentiated only if the reaction of one differs significantly from the reaction of the other on the same variety under the same conditions. The finer differences of reaction usually given in a final race analysis have therefore been ignored in this survey, and the terms immune, resistant, and susceptible adopted. Immunity and susceptibility are probably synonymous with complete incompatibility and complete compatibility respectively between host and pathogen. Resistance includes the intermediate range of reaction probably synonymous with intermediate incompatibility. The use of the term seems quite valid, since such reactions less than completely susceptible generally agree with field resistance.

Reaction Levels of the Differential Varieties.

The following is a brief summary of the number of reaction levels determined for each of the differential varieties.

Buda: 2: susceptible and a highly variable resistant reaction.

Williston Golden: 2: susceptible and resistant. The resistant reaction tended towards immunity and was characterized by necrosis and a variable number of pustules rarely exceeding Flor's type "3–". A slight difference in reaction between races 1 and 17 was too fine to justify differentiation of two intermediate reactions.

AKMOLINSK: 2: susceptible and resistant. The latter approximated fairly closely to Flor's type "1" reaction The susceptible reaction to race 1 may have been slightly lower in the scale of susceptibility than that evoked by other races. The variety appeared to have a measure of resistance to Punjab-attacking race 1 later in development, but in the seedling stage under optimal growing conditions it was fully susceptible.

J.W.S.: 3: susceptible, resistant, immune. The resistant reaction to race 1 ranged from almost complete susceptibility to near immunity according to the conditions of incubation and the inoculation technique used.

ABYSSINIAN: 2: susceptible and immune. The susceptible reaction was sometimes depressed slightly to moderate susceptibility.

KENYA: 3: susceptible, resistant, and immune. The resistant reaction was characterized by necrosis, type "2" and occasional type "3–" pustules. The susceptible reaction was sometimes reduced to moderate susceptibility.

ARGENTINE 705-1: 2: a highly variable resistant reaction varying from near immunity to near susceptibility, and a rather variable susceptible reaction commonly depressed to moderate susceptibility.

VERY PALE BLUE CRIMPED: 3: susceptible, resistant, and immune. The resistant reaction ranged from near immunity to type "2" reaction. The susceptible reaction was most commonly reduced to moderate susceptibility. It was very subject to variations in incubation conditions.

OTTAWA 770B: 2: susceptible and immune. Two very stable reactions.

ARGENTINE 462: 1: immune.

BISON: 2: full susceptibility, immune. Both reactions were extremely stable.

PUNJAB: 2: susceptible and immune. The susceptible reaction to the only race to which it succumbed was one of the most virulent noted during these studies.

WALSH: 2: susceptible and resistant. The reaction to race 1 approximated more closely to immunity than that given by other non-Walsh-attacking races. The resistant reaction to these latter races was characterized by severe necrosis, with occasional type "2" pustules. The susceptible reaction was rather low in the scale of susceptibility.

MORYE: 1: immune.

NEWLAND: 2: susceptible and immune. Several races gave a delayed resistant reaction some time after completion of the usual incubation period. It was characterized by isolated and never abundant type "3" pustules. There was no necrosis. Uredospores taken from these pustules and built up on fully susceptible F257 failed to induce a more susceptible reaction. The pustules did not represent contaminant races. The races evoking this reaction more commonly gave complete immune reactions. The sparsity of type "3" pustules and the inconsistent development of the reaction did not justify differentiation of a resistant reaction level.

BOLLEY GOLDEN: 3: susceptible, resistant, and immune. The resistant reaction agreed with Flor's type "1", but was often depressed to immunity. Small isolated non-necrotic pustules like those recorded for Newland were sometimes associated with this reaction.

ITALIA ROMA: 2: susceptible and immune. There seemed to be a resistant reaction, but it was not sufficiently consistent to differentiate it from the slightly variable immune reaction. The susceptible reaction was quite commonly depressed to moderate susceptibility and occasionally to a resistant reaction.

LEONA: 2: susceptible, sometimes depressed to moderate susceptibility and a slightly variable immune reaction.

TAMMES' PALE BLUE: 2: susceptible and immune. Some immune reactions developed occasional type "3" pustules rather like those reported for Newland and Bolley Golden. The susceptible reaction varied about a norm of moderate susceptibility.

KOTO: 1: immune.

TABLE 1.

Details of the Virulence of Races Identified During the Survey on the Differential Varieties Designated by University Fx Accession Number.

Race.	1 Bu.	2 WG.	3 Ak.	4 JWS.	5 Ab.	6 Ke.	7 Arg.	9 PBC.	10 Ot.	11 Arg.	13 Bi.	14 Pu.	86 Wa.	318 Mo.	319 Ne.	320 BG.	321 IR.	322 Le.	323 TPB.	326 Ko.
1	R	R	S	R	I	I	R	I	I	I	I	S	R	I	I	I	I	I	I	I
2	S	S	R	I	I	R	R	S	I	I	S	I	R	I	I	I	I	I	I	I
3	S	S	S	S	S	S	R	S	I	I	S	I	R	I	I	R	S	S	I	I
4	S	S	R	S	I	S	R	S	I	I	S	I	R	I	S	S	S	I	I	I
5	S	S	S	S	S	S	S	S	I	I	S	I	R	I	S	I	I	S	I	I
6	S	S	S	S	S	R	S	S	I	I	S	I	R	I	I	I	I	S	I	I
7	S	S	S	S	S	S	S	S	S	I	S	I	R	I	I	I	I	S	I	I
8	S	S	S	S	S	R	R	R	I	I	S	I	R	I	I	I	I	S	I	I
9	S	S	S	S	S	R	S	S	I	I	S	I	R	I	S	I	I	S	I	I
10	S	S	S	S	S	S	S	S	I	I	S	I	R	I	I	I	I	S	I	I
11	S	S	S	S	S	S	S	S	I	I	S	I	S	I	I	I	I	S	I	I
12	S	S	S	S	S	S	S	R	I	I	S	I	R	I	I	S	S	S	I	I
13	S	S	S	S	S	S	S	S	I	I	S	I	R	I	S	S	S	S	I	I
14	S	S	S	S	S	S	S	S	S	I	S	I	R	I	S	I	I	S	I	I
15	S	S	S	S	S	R	S	S	S	I	S	I	R	I	I	I	I	S	I	I
16	S	S	S	S	S	S	S	S	S	I	S	I	R	I	I	R	S	S	I	I
17	S	R	S	S	S	S	S	S	S	I	S	I	R	I	I	I	I	I	S	I
18	S	S	S	S	S	R	S	R	I	I	S	I	R	I	I	I	I	S	I	I

I = Immune. R = Resistant. S = Susceptible.

Races Identified During the Physiologic Race Survey.

On the basis of the reaction levels determined for each variety eighteen races were detected among sixty-two accessions collected in the field. These races were numerically designated, instead of following the alphabetical system previously adopted by Waterhouse and Watson (1943). This was considered advisable for three reasons. The frequency of occurrence of new races would soon have exhausted the reservoir of English alphabet symbols. The differential series adopted in the current survey included two varieties not used in any prior survey. The reaction given by Buda (used in previous surveys) to Punjab-attacking accessions was not considered sufficiently reliable to justify separation of races on this variety. The last two points complicated comparison of races differentiated in this and earlier surveys. A new system was therefore adopted to avoid confusion with results obtained in earlier surveys.

The reaction of each race on the differential series is listed in Table 1. A key for the identification of each race was also devised (Table 2).

TABLE 2.

Key for the Identification of Australian Races of Melampsora lini *Determined in the Current Survey.*

	Race
Punjab: Susceptible	1
Punjab: Immune.	
Ottawa 770B: Immune.	
Walsh: Susceptible	11
Walsh: Resistant.	
Newland: Immune.	
Very Pale Blue Crimped: Resistant.	
Argentine 705–1: Resistant	8
Argentine 705–1: Susceptible.	
Kenya: Resistant	18
Kenya: Susceptible	12
Very Pale Blue Crimped: Susceptible.	
Abyssinian: Immune	2
Abyssinian: Susceptible.	
Bolley Golden: Immune	6
Bolley Golden: Resistant.	
Argentine 705–1: Resistant	3
Argentine 705–1: Susceptible	10
Newland: Susceptible.	
Abyssinian: Immune	4
Abyssinian: Susceptible.	
Bolley Golden: Immune.	
Kenya: Resistant	9
Kenya: Susceptible	5
Bolley Golden: Susceptible	13
Ottawa 770B: Susceptible.	
Newland: Immune.	
Williston Golden: Resistant	17
Williston Golden: Susceptible.	
Kenya: Resistant	15
Kenya: Susceptible.	
Italia Roma: Immune	7
Italia Roma: Susceptible	16
Newland: Susceptible	14

Comparison of Races Identified During the Current Survey with Races Identified Earlier.

Comparison of races identified in the present survey with those identified by previous workers was complicated by slight differences in the varieties used and by differences in the system of reaction classification. Four different reactions were assigned to Kenya, Argentine 705-1, and Very Pale Blue Crimped by other workers. This was considered an unjustifiably fine distinction of reactions during this survey, and only three reactions were assigned to Kenya, and two to each of the last two varieties. Despite this, there was sufficient common ground for comparison.

Comparison with results obtained in the first survey by Waterhouse and Watson in 1941 and 1943 suggested a marked change in the rust population in Victoria and Western Australia. A more intensive survey would probably have revealed the same situation in South Australia.

Eight of the twelve races identified from Western Australia since 1948 were distinctly different from races detected between 1940 and 1942. Among them five new races could be designated even on the restricted differential series used by Waterhouse and Watson. Races 2 and 4 were more avirulent on the original differential series used in Survey I than earlier non-Punjab-attacking races, and races 7, 14, 15, 16 and 17 were highly virulent on Ottawa 770B, immune to all Australian races identified during the earlier survey.

Six of the thirteen races identified in Victoria since 1948 were distinctly different from races identified in Survey I. Each of the six could be differentiated from each other on the original Survey I differential series. Races 2 and 4 were less virulent than non-Punjab-attacking races identified in Survey I. Races 8, 12 and 18 differed from Survey I races in their resistant reaction on Very Pale Blue Crimped, and race 14 was the first Ottawa 770B-attacking race recorded in the State.

Fewer collections were received from South Australia, and correspondingly fewer races were identified. But one of the five identified since 1948, race 2, differed from the four races recorded in 1943.

The race position remained unchanged in New South Wales, where both collections received were Punjab-attacking.

No collections were received from Queensland prior to 1948. The seven received and analysed since then were identified as race 1, a Punjab-attacking race.

A marked shift in the race population was noted when races identified by Watson, Charles, and Kerr between 1943 and 1948 were compared with races identified in the current survey. Prior to the current survey, only three Newland-attacking races had been identified. There were no Bolley Golden or Ottawa 770B-attacking races, and Walsh was relatively free from infection in the field until 1948. This survey identified six Newland-attacking races, 4, 5, 9, 12, 13 and 14, each of them apparently different from already determined Newland-attacking races. Five Ottawa 770B-attacking races, 7, 14, 15, 16 and 17, and three Bolley Golden-attacking races, 4, 12 and 13, were detected. An important Walsh-attacking race, race 11, was discovered in Victoria.

Only four of the races identified in this survey bore any close resemblance to races identified by previous surveys. Races 6 and 10 agreed fairly closely with Charles' races C and E respectively. Race 2 was identical with race K. The latter race was identified by Kerr in 1948. It was later found to give slightly different reactions on J.W.S., Akmolinsk, Kenya, and Argentine 705–1 than those listed by Millikan (1951). Race 1 was probably a composite of races A and F, since the reaction of Buda was not used to separate races in this survey as it had been to differentiate races A and F in earlier surveys.

Races A and F were differentiated by their reaction on Buda, and since this variety was rejected during the current survey, differentiation between races A and F was impossible. Race 1 cannot be equated with either of the former races and must be equated with both. Its reaction differs somewhat from that indicated for races A and F on two of the differential hosts. This could be attributed to the inoculation technique adopted in this survey. This induced a more virulent reaction than the spore dusting method of earlier surveys. When race 1 was inoculated onto J.W.S. and Akmolinsk by the latter method it gave virtually the same reactions, immune and resistant respectively, as those noted by Waterhouse and Watson for races A and F for these two varieties.

It was concluded that fourteen of the eighteen races identified in this survey had not been recorded before in Australia, namely races 3, 4, 5, 7, 8, 9, 11, 12, 13, 14, 15, 16, 17, and 18. Some of these races may have been present in the field when earlier

surveys were carried out, but may have been overlooked because insufficient districts were sampled, or because they constituted only a very small part of the total rust population at that time. But the very high percentage of new races among those identified in the current survey suggests a marked change in the race complex since the first surveys were carried out.

TABLE 3.

Distribution of Australian Races in Time and State.

State.	Year.	Race.																	Total.
		1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	
Western Australia.	1948	—	—	—	1	2	—	—	—	—	—	—	—	—	—	—	—	—	1	4
	1949	—	1	—	—	—	—	1	—	—	—	—	—	—	—	—	—	—	—	2
	1950	1	—	—	—	—	—	—	—	1	—	—	—	1	2	—	—	—	—	5
	1951	—	—	—	—	3	—	—	—	—	—	—	—	2	—	1	1	1	—	8
South Australia.	1948	—	1	1	—	1	—	—	—	—	—	—	—	—	—	—	—	—	—	3
	1950	1	—	—	—	—	—	—	—	—	1	—	—	—	—	—	—	—	—	2
	1951	1	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2
	1952	2	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	2
Victoria.	1948	—	1	—	—	2	1	—	—	—	—	—	—	—	—	—	—	—	—	4
	1949	1	2	1	1	1	1	—	2	1	—	1	—	—	—	—	—	—	—	11
	1950	—	—	—	1	—	—	—	—	—	1	—	1	—	—	—	—	—	—	3
	1951	—	—	—	—	—	—	—	—	—	1	—	—	—	—	—	—	—	1	2
	1952	—	—	2	—	—	—	—	—	—	2	—	—	—	1	—	—	—	—	5
Queensland.	1948	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1
	1952	6	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	6
New South Wales.	1949	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1
	1952	1	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	—	1
Total of each race	..	15	6	4	3	9	2	1	2	2	5	1	1	3	3	1	1	1	2	62

Races Detected in Each State During the Survey.

Queensland .. .. Race 1.
New South Wales .. .. Race 1.
Victoria .. .. .. Races 1, 2, 3, 4, 5, 6, 8, 9, 10, 11, 12, 14, 18.
South Australia .. .. Races 1, 2, 3, 5, 10.
Western Australia .. .. Races 1, 2, 4, 5, 7, 9, 13, 14, 15, 16, 17, 18.

Geographic Distribution of Each Race and the Major Race Groups.

The distribution of the eighteen races according to the year of collection and State is given in Table 3. The districts in each State in which each race was found and a summary of the race complex of each State are listed in Table 4.

There was a remarkable diversity of non-Punjab-attacking races in the flax-growing southern States, and a complete absence of any of these races in the eastern linseed-growing States, New South Wales and Queensland.

There was no one dominant race among the seventeen non-Punjab-attacking races found in the south, although races 2, 5 and 10 occurred with a slightly greater frequency than the others. This follows the pattern of Flor's findings in the United States. Only two non-Punjab-attacking races, 2 and 5, occurred in all three southern States. Nine races were restricted to a single State.

Newland-attacking races were widely distributed in the southern belt during this survey, but were already established before the survey. Ottawa 770B-attacking races

by contrast were recorded in Australia for the first time since this survey began. There was no trace of such a race among the four hundred or more collections analysed previously. In 1948 Western Australian departmental officers reported sparse infection of Ottawa 770B seedlings in a small experimental plot at Kojunup. They attributed it to varietal impurity. The first Ottawa-attacking race, race 7, was identified in 1949 in a collection from Boyup Brook. Another Ottawa-attacking race, race 14, was identified from the same district in 1950. Three other Ottawa-attacking races, races 15, 16 and 17, were discovered in 1951 in the same area at North Boyup Brook, Newbicup, and Kulikup respectively. No further collections were received from the State, but a member of the local Department of Agriculture wrote that "The

TABLE 4.

Details of the State and District of Occurrence of the Races Determined during this Survey.

Race 1.	Queensland: Gatton 1948, Hermitage and Toowoomba 1952, Biloela 1950.*
	N.S.W.: Castle Hill 1949, Tibooburra 1952, Curlewis 1950.*
	Victoria: Lake Bolac 1949, Werribee 1953.*
	South Australia: Waite Institute 1950, 1952, Corry Point 1951.
	Western Australia: Boyup Brook 1950.
Race 2.	Victoria: Lake Bolac 1948, Winchelsea, Colac 1949.
	South Australia: Laura 1948, Waite Institute 1951.
	Western Australia: Boyup Brook 1949.
Race 3.	Victoria: Winchelsea 1949, Thorpdale 1952.
	South Australia: Maclaren Vale 1948.
Race 4.	Victoria: Werribee 1949, 1950.
	Western Australia: Mayanup 1948.
Race 5.	Victoria: Myrtleford 1948, Lake Bolac 1948, 1949.
	South Australia: Undalaya, 1948.
	Western Australia: Kojunup 1948, North Boyup Brook, Benjinup 1951.
Race 6.	Victoria: Colac 1948, Winchelsea 1949.
Race 7.	Western Australia: Boyup Brook 1949.
Race 8.	Victoria: Lismore, Casterton 1949.
Race 9.	Victoria: Winchelsea 1949.
	Western Australia: Boyup Brook 1950.
Race 10.	Victoria: Colac 1950, Drouin 1951, Thorpdale 1952.
	South Australia: Laura 1950.
Race 11.	Victoria: Casterton 1949.
Race 12.	Victoria: Birregurra 1950.
Race 13.	Western Australia: Boyup Brook 1950, Mayanup 1951.
Race 14.	Victoria: Thorpdale 1952.
	Western Australia: Boyup Brook 1950.
Race 15.	Western Australia: North Boyup Brook 1951.
Race 16.	Western Australia: Newbicup 1951.
Race 17.	Western Australia: Kulikup 1951.
Race 18.	Western Australia: Kojunup 1948.
	Victoria: Lismore 1951.

* Not positively identified as race 1 at Sydney University, since no viable inoculum was received, but definitely Punjab-attacking races, and therefore closely related to race 1, if not actually race 1.

Ottawa-attacking race (group) was very active (in 1952), and will, I think, considerably lower the fibre value of commercial crops of Wada". It seemed to have supplanted the old races. Until then the race group had been confined to Western Australia, but in early December, 1952, race 14 was identified on a specimen of Rust Resistant Norfolk Earl growing in Government experimental plots at Thorpdale, Victoria.

Field infection of Walsh, the only commercially grown linseed variety in Australia, was relatively insignificant until 1948. Although Walsh had not yet been included in the differential series, it is fairly certain from the above observation that no major Walsh-attacking race was then present. Race 11 was discovered in a collection from Casterton, Victoria, in 1949. It was highly virulent on the rather mixed commercial variety, attacking 70% of the seedlings in glasshouse tests at Sydney. The single plant selection, previously resistant to already identified Australian races, was fully susceptible. Despite its virulence and an abundance of susceptible host material in the field, it did not appear to have spread. But since the conclusion of this survey

there has been a report of a new Walsh-attacking race at Thorpdale and Casterton (Debrett, 1954).

Bolley Golden-attacking races were found in Victoria and Western Australia. Race 17, already mentioned as an Ottawa-attacking race, was the first occurrence of a Tammes' Pale Blue-attacking race in Australia. It was located at Kulikup in Western Australia.

Race 1 was the only Punjab-attacking race identified during the survey. It was the most common of the eighteen races and was detected in fifteen of the sixty-two collections. It occurred in all five States from which the collections were received. It was most common in Queensland. It was less common in New South Wales, Victoria, South Australia and Western Australia. But there was ample evidence from information supplied by officers of the State departments of agriculture and in the literature to indicate a more widespread occurrence.

Two Punjab-attacking accessions from New South Wales were identified as race 1. One was obtained from Tibooburra on wild flax, *L. marginale*. The other was obtained from linseed experimental plots at Castle Hill, near Sydney. Both districts were hundreds of miles from any known source of infection from commercial varieties of flax or linseed. Race 1 or a closely related race was also present in experimental plots at Curlewis in the north-western wheat belt. Several flax and linseed varieties were sown at the University Experiment Station, Curlewis, in May, 1950. The seed was disease-free, and there was no known source of rust inoculum in any of the commercial crops grown within 500 miles of the district. Punjab and Imperial, which in tests at Sydney University were immune to every race identified in this survey except race 1, were heavily infected by the end of September. The spores received on specimens sent from the district were inviable on receipt, but the race was almost certainly race 1 or a closely related race. The other varieties, including Concurrent, were uninfected. Since this variety was fully susceptible to all the non-Punjab-attacking races identified during this and previous surveys the immunity of this variety at Curlewis was strong proof of the absence of non-Punjab-attacking races from the district.

Rust was also noted on *L. marginale* at Pucuwan, 11 miles from Temora in the south-western wheat belt, during a field trip in November, 1948. Mr. Evans, of the Sydney University Botany Department, also reported that rust was quite common on the species near Sydney. Since twelve of thirteen collections received from the species since 1941 were Punjab-attacking races it is reasonable to infer that Punjab-attacking races were present near Pucuwan in 1948 and are common near Sydney in most years.

Only one collection of race 1 was received from Victoria during this survey, but seed of the differential varieties and F257 was sent to Mr. P. Debrett, of the Victorian Department of Agriculture, in 1952. Owing to excessive rain he was unable to make sowings in the Werribee district until late in the year. Punjab became heavily infected during February. The other varieties were immune. Since no inoculum was forwarded, the exact nature of the race is unknown. But since it was a Punjab-attacking race, it was probably race 1 or a closely related race. Since Bison and F257 were not infected, none of the common non-Punjab-attacking races could have been present in the field at that time of the year.

Four collections of race 1 were received from South Australia, one from *L. marginale* growing at Corry Point in 1951, and three from Punjab-type Indian linseed varieties growing at Waite Agricultural Institute. The race was present each year from 1950 to 1952 and constituted four of the ten collections received from the State.

One collection of race 1 was received from Western Australia. It was found as a mixture with race 14 on supposedly Concurrent plants growing at Boyup Brook in 1950. It must have been more common, since fifteen of twenty-eight rust collections sent in from the State in 1942 were identified as race A by Waterhouse and Watson.

The first Punjab-attacking collection from Queensland was collected from linseed growing at Gatton in 1948. No further collections were received from the State until

1952, but during that year special efforts were made to determine whether race 1 was the only race present in Queensland.

Records were kindly forwarded by the Plant Introduction Officer of the Commonwealth Scientific and Industrial Research Organization at the Cooper Laboratory, Lawes, Queensland, giving the mean intensity of rust infection in this district of the differential varieties used by Waterhouse and Watson and of Liral Crown and Concurrent. Results obtained in 1948 and 1949 showed that only Punjab-attacking races were present. Tests were discontinued in later years, but up to the end of this survey there was no record of infection of varieties such as Bison and Concurrent, known to be susceptible to all of the non-Punjab-attacking races of the southern States.

Seed of the varieties Punjab (susceptible only to race 1) and F257 (susceptible to all the races, including race 1) was distributed to experimental stations at Warwick, Hermitage, Toowoomba, and Kingaroy in 1952. They were grown under conditions most likely to induce infection, but remained uninfected at Kingaroy and Warwick. Infection of both varieties was reported at Hermitage in mid-November. The race

TABLE 5.

Distribution of Major Race Groups in Australia.

Punjab-attacking Race Group: Race 1.
Queensland: Gatton, Hermitage, Toowoomba, Biloela.
N.S.W.: Castle Hill, Tibooburra, Curlewis.
Victoria: Lake Bolac, Werribee.
South Australia: Waite Institute (Adelaide), Corry Point.
Western Australia: Boyup Brook.

Ottawa 770B-attacking Races: 7, 14, 15, 16 and 17.
Victoria: Thorpdale.
Western Australia: Boyup Brook, North Boyup Brook, Kulikup, Newbicup.

Newland-attacking Races: 4, 5, 9, 12, 13 and 14.
Victoria: Winchelsea, Lake Bolac, Birregurra, Werribee, Myrtleford, Thorpdale.
South Australia: Undalaya.
Western Australia: Mayanup, Kojunup, North Boyup Brook, Benjinup, Boyup Brook.

Bolley Golden-attacking Races: 4, 12 and 13.
Victoria: Birregurra, Werribee.
Western Australia: Mayanup, Boyup Brook.

Walsh-attacking Race: 11.
Victoria: Casterton.

was not identified, but three collections received from the same district between September and October were identified as race 1.

The varieties were sown at three localities in the Toowoomba district. Sowings were made at regular intervals and a close check kept on each plot. No rust was recorded at one of the locations. Both varieties were infected in early December at the other locations. F257 was moderately infected. Punjab was heavily infected. The rust was identified as race 1. Sowings were made on 6th January, 1953, and at fortnightly intervals for some time afterwards. Every attempt was made to induce healthy succulent growth to encourage infection, but no rust was recorded on either variety after December.

Rust has appeared at Biloela Experiment Station, 560 miles north of Brisbane. This was almost certainly race 1 or a related race, since rust specimens sent to Sydney from the area in October, 1950, were from Punjab and Imperial.

Notes on the Cultivated and Wild Hosts in Australia.

Concurrent was highly susceptible in the seedling stage to all the races identified during the survey. It was somewhat less susceptible to race 1 than the other races in the seedling stage, and infection was usually restricted to the leaves, rarely spreading to the stems. Later in maturity it seemed to acquire complete immunity to the Punjab-attacking races. The same mature plant resistance seemed to be effective against the new highly virulent Ottawa-attacking races of Western Australia. This was not apparent until 1951. The first Ottawa-attacking race, race 7, was

collected at Boyup Brook from the variety Boyup in 1949. The following year race 14 was identified from two collections of Concurrent. Eight collections were received from neighbouring areas in 1951. The three collections from Concurrent yielded non-Ottawa-attacking races. Three different Ottawa-attacking races were isolated from the Wada specimens. This suggested that Concurrent was resistant to the most recent Ottawa-attacking races. The Western Australian Department of Agriculture late in 1952 reported that Concurrent was then free of rust although Wada was seriously affected.

Excised shoots of Concurrent were inoculated and incubated with an Ottawa-attacking race, race 17, and a non-Ottawa-attacking race, race 6. The two sets of shoots were kept under identical conditions in the same part of the glasshouse until sporulation and for several weeks afterwards. Both sets of shoots became heavily infected within two weeks. Shortly afterwards the race 6 infection spread to the stems, and subsequent shoot growth was stunted. Race 17 infection was restricted to the leaves, and the shoots recovered and continued vigorous growth (Plate iv, 2).

TABLE 6.

Races Isolated from the Varieties and Species Listed Below.

Host.	Races.
Linum usitatissimum:	
Concurrent	1, 4, 5, 13, 14.
Liral Crown	2, 3, 5, 8, 10.
Walsh	3, 6, 8, 11, 12, 18.
Wada	9, 10, 13, 15, 16, 17.
Boyup (A3)	3, 5, 7, 13.
Punjab, Imperial	1.
Unidentified flax varieties, probably Concurrent or Liral Crown	2, 4, 5, 10, 18.
Linum marginale	1.

Wada and Boyup were developed as rust-resistant varieties to replace Concurrent and Liral Crown. Tests at Sydney University showed that both varieties carried an appreciable percentage of off-type plants susceptible to most of the non-Punjab-attacking races with which they were tested. Both were fully susceptible in the seedling stage to the Ottawa-attacking races. But Boyup, like Concurrent, from which it is reported to have derived by natural crossing with Wada, seemed to have mature plant resistance to these races in the field. The two collections received from Boyup, Western Australia, in 1951 yielded non-Ottawa-attacking races, and a letter from the Department of Agriculture in November of 1952 stated that it was not as badly infected as Wada in the field.

The commercial variety Walsh contained up to 20% seedlings susceptible to many of the non-Punjab-attacking races. The occurrence of these susceptible plants probably gave rise to the many reports of important new Walsh-attacking races from the southern flax-growing districts. Race 11 was the only race to which most Walsh seedlings were fully susceptible, but eight of twenty-four single plant selections from the commercial variety were immune to this race. The occurrence of race 11 at Casterton in 1949 posed a considerable threat to the linseed-growing industry, but up to 1952 no further specimens of this race were received from Victoria. A report from Victoria in 1954 would seem to indicate the appearance of a new Walsh-attacking race in the Casterton and Thorpdale districts.

Linum marginale is probably an important host of *M. lini* in Australia. It is native to Australia and has been a host to *M. lini* since the first recorded observations of rust in the country. (The species is common in New Zealand, where it is regarded as an important carry-over host, particularly during winter. New Zealand crops are spring sown. Unlike Australia, there is an abundance of cultivated host material during summer, but a dearth of the host during winter.) It was reported from

Western Australia: "The evidence is clear in this State that *L. marginale* is an important hold over host for the rust fungus." The species is quite common in Victoria and South Australia. It has been found at such widely separated places in New South Wales as Sydney, Bourke, Curlewis, Tibooburra and Armidale.

It appeared to be much more restricted in its occurrence in Queensland. One report indicated that it had never been seen in the Lawes district or on the Darling Downs. The Government Botanist reported that the species was not common and the four localities from which specimens had been received were in the southern portions of the State towards the New South Wales border.

The species is not homogeneous in its reaction to *M. lini*. During the course of these studies, seed of the species was received from several States. The different lines were sown and tested for reaction to race 1 and several of the non-Punjab-attacking races. The lines tested were immune to several accessions of race 1 and also races 2, 6 and 7. Other lines, however, have been susceptible to race 1.

Factors Affecting the Incidence of the Rust in the Field.

The yearly and seasonal incidence of rust was directly correlated with the availability of susceptible material. Flax acreages have diminished steadily since the last war from 61,000 acres in 1944 to approximately 12,000 acres in 1948, and approximately 4,800 in 1950. This was paralleled by a reduction in the number of collections of rust received from the field.

Most of the crops were harvested by the end of December, leaving a sparse supply of volunteer plants to carry the rust over the hot summer months of January and February in the uredospore stage. This partly accounts for the fact that few samples of rust were received from the field later than December. Details of the earliest and latest collections of rust received from the field are given in Table 7. The dates given

TABLE 7.

Details of the Earliest and Latest Collections of Rust Received from Each State.
1948-1952.

State.	Earliest Collection.	Latest Collection.
Queensland	8th September.	13th December.
New South Wales	26th September.	22nd November.
Victoria	3rd August.	23rd December.
South Australia	9th October.	15th January.
Western Australia	10th September.	4th December.

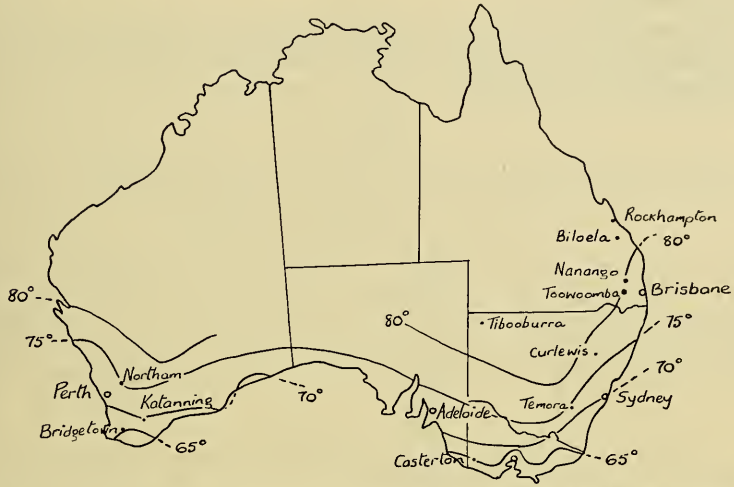
in this table do not indicate the earliest or latest appearances of *M. lini* in the field in the uredospore stage, but do help to indicate roughly the periods of maximum development of the rust in the uredospore stage.

The rust is evidently most abundant in the field in the uredospore stage from early September to December. Lack of rust before this period can be attributed to low field temperatures.

Observations in the field and in the glasshouse at Sydney and reports from workers in other States indicate that Punjab-attacking race 1 is better able to survive higher summer temperatures than the non-Punjab-attacking races in the uredospore stage. Non-Punjab-attacking race 2 was unable to survive summer temperatures in the field at Sydney. But Punjab-attacking race 1 survived without difficulty and caused severe infection.

The high temperature tolerance of Punjab-attacking races was also apparent from Mr. P. Debrett's report on the infection of varieties forwarded to him at Werribee in 1952. Punjab was severely infected during February, 1953. Non-Punjab-attacking races were much more common than Punjab-attacking races in his district up to December

in earlier years. But they were unable to maintain themselves in the uredospore stage during late summer, since Bison and F257, highly susceptible to these races, were uninfected during February.



Text-fig. 2.—Climatic map of Australia indicating mid-summer isotherms of flax and linseed growing States.

Approximately 1,000 miles further north, at Toowoomba, even Punjab-attacking races were less able to survive summer in the uredospore stage. Race 1 was present in the field until late December but failed to cause infection of susceptible material during January and February.

TABLE 8.

Summer and Winter Temperatures of Potential Flax Growing Districts in Australia.

State.	District.	Mean Temperature during Mid Winter. °F.	Mean Temperature during Summer. °F.
Tasmania.	Launceston	47	64
Western Australia	Bunbury	55	73 Dec.–Jan.
	Katanning	50	70 Dec.–Jan.
	York	50	76 Dec.–Jan.
South Australia.	Waite Institute ..	50	70 Dec.–Feb.
	Millicent, Kapunda ..	Much the same as Waite Institute.	
	Mt. Barker, Clare ..		
Victoria.	Colac	43	64 Dec.–Feb.
	Leongatha	44	64 Dec.–Feb.
	Wangaratta	44	72 Dec.–Feb.
	Myrtleford	38	70 Dec.–Jan.
New South Wales	Albury	44	73 Dec.–Feb.
	Cootamundra	38	72 Dec.–Feb.
	Glen Innes	38	68 Dec.–Jan.
	Inverell	44	73 Dec.–Jan.
	Bathurst	38	68 Dec.–Jan.

Although non-Punjab-attacking races lacked tolerance to high temperature they were able to survive in the field in the uredospore stage in some districts in the southern States when the host was available. Rusted Walsh plants were received

from Mount Gambier in South Australia in mid-January, 1953. The rust was inviable on receipt. But since Walsh is completely immune to race 1, the only Punjab-attacking race identified during this survey, it is safe to attribute the infection in this instance to a non-Punjab-attacking race.

Since summer temperatures played an important role in regulating the seasonal incidence of rust, efforts were made to determine summer temperatures of the flax- and linseed-growing areas of Australia.

TABLE 9.

Summer Temperatures in Western Australia Flax Growing Districts.

Month.	Average of 40 years. °F.				
	Bridgetown.			Katanning. Mean.	York. Mean.
	Mean.	Maximum.	Hottest Day.		
November	61·9	76·4	103	63·7	67·8
December	66·4	82·3	105	68·5	73·7
January	69·1	85·5	109	71·1	76·4
February	68·6	85·3	115	70·6	76·1
March	65·3	80·4	105	66·8	71·7

Climatic graphs prepared by Dr. Forster (1941) to determine suitable areas for flax production in Australia yielded the information found in Table 8. Additional information was furnished by the Western Australian Department of Agriculture for Western Australian flax areas (Table 9). Temperatures at representative areas on the Darling Downs and other districts in Queensland were obtained from S. G. Gray's report on variety trials, 1950 (Table 10).

These figures agreed with the climate maps indicating the major temperature zones in Australia during the summer. It will be noticed that none of the Queensland districts had a mean temperature during January of less than 71°F. Some at least of the flax-growing districts of the southern States had a mean temperature during January of 70°F. or less. This fact is highlighted by the January (mid-summer) isotherms. Most of the southern flax-growing areas of Victoria, South Australia and Western Australia lie in a moderately temperate zone by comparison with the linseed-growing areas of north-west New South Wales and Queensland.

TABLE 10.

Mid Summer and Mid Winter Temperatures in Queensland Linseed Growing Districts. °F.

Month.	Mean Monthly Temperature.				
	Lawes.	Toowoomba.	Pittsworth.	Killarney.	Nanango.
January	77·2	71·2	74·1	71·9	74·1
July	56·1	50·7	51·4	59·0	51·2

Additional Comments on the Virulence of Punjab-Attacking and Non-Punjab-Attacking Races.

The results already summarized indicate an important division within the *M. lini* complex between the Punjab-attacking and the non-Punjab-attacking races. Observations not directly relevant to the survey were made during the course of these studies, which confirmed the difference between the pathogenicity of the two race groups.

It was possible within the reaction level of susceptibility to distinguish varying levels of virulence. Such distinctions could be readily defined by noting the following.

1. The extent of infection. In cases of milder susceptibility the uredosori were entirely or almost entirely restricted to the leaves, seldom spreading to the stems. In cases of extreme susceptibility the uredosori developed as vigorously on the stems as on the leaves.

2. The extent of plant recovery. In cases of mild susceptibility the inoculated plants commonly recovered and resumed normal growth unless deliberate attempts were made to induce further infection. In cases of extreme susceptibility plant growth was severely retarded or completely checked. Natural secondary infection of new growth was common.

3. Varieties showing the most severe susceptible reactions commonly developed heavy infection by the spore dusting technique. Varieties giving a milder reaction developed a very sparse infection by the spore dusting method.

Without exception non-Punjab-attacking races evoked a maximally virulent susceptible reaction on the variety F257. This variety was also susceptible to race 1, but invariably gave a less virulent susceptible reaction. A quantitative assessment of the difference in virulence of the non-Punjab-attacking and the Punjab-attacking races on F257 is summarized in Table 11. With the majority of the seedlings inoculated

TABLE 11.

Stem Infection of Pot Grown Seedlings Inoculated 7th May, 1953. Read 29th May, 1953.

Race.	Variety.	Number of Stems with Uredosori.		
		Heavy Infection.	Very Mild Infection.	No Infection.
1	F257	2	18	—
1	Punjab	19	—	4
6	F257	12	—	5
2	F257	10	—	5

with NPA races infection spread to the stem, producing prominent uredosori from which considerable quantities of inoculum could be drawn. Only two of the twenty seedlings inoculated with race 1 developed many stem uredosori. Eight stems were scarcely infected, and ten stems developed only a few scattered pustules. Race 1, however, gave a reaction of maximum virulence on Punjab seedlings, nineteen of twenty-three seedlings developing a heavy crop of uredosori on the stem.

In another test with excised shoots the milder virulence of race 1 on F257 was reflected in the almost complete lack of natural secondary infection after sporulation of the primary infection. Only one shoot showed any appreciable secondary infection. Only six secondary pustules developed on the other twenty-five shoots. The extreme virulence of the same race on Punjab resulted in heavy secondary infection of twenty-one of the thirty-four shoots.

Spore dusting bulk inoculation resulted in heavy infection of Punjab plants dusted with uredospores of race 1, placed in a moisture chamber and sprayed at intervals. F257 plants treated simultaneously in the same chamber developed only a light infection (Plate iv, 1 and 3).

The susceptibility of Punjab to race 1 was one of the most virulent reactions noted during the course of these studies. Punjab-attacking races from New Zealand induced a much milder reaction. Australian race 1 and New Zealand Punjab-attacking races 3 and 13 were cultured on Punjab excised shoots. The Australian race 1 accessions 507, 610, 613 and 624 from different sources in Queensland, and accession 621 from South Australia induced a maximum susceptible reaction with typical type 4 pustules. Infection spread to the stems; the plants became stunted and did not resume normal growth. Shoots inoculated with the New Zealand races developed type 3 pustules.

After three to four weeks the infected leaves died and normal growth resumed. Infection did not spread to the stems (Table 12).

TABLE 12.

Comparison of Virulence of Australian and New Zealand Punjab-attacking Races.

	Race 1 Accessions.					New Zealand Races.	
	507	610	613	621	624	3	13
Stems with uredosori ..	51/53	9/9	8/8	11/11	7/9	0/18	0/19

A NOTE ON UREDOSPORE MORPHOLOGY.

Measurements were made of uredospores of races 1, 2, 6 and 7 over a period of two weeks. The spores were collected from susceptible plants cultured under identical conditions and stored at 0° to 2°C. in the refrigerator for no more than four weeks. The spores were prepared for measurement by dusting onto a drop of specially prepared lacto-phenol on a thoroughly clean microscope slide. They were covered lightly with coverslips and checked after at least half an hour under the oil immersion objective. Provided all excess lacto-phenol was blotted up and the immersion oil was very fluid the coverslip remained firmly fixed to the slide as the slide was moved under the objective. The tube of the microscope was extended until each division on the scale in the eyepiece corresponded to 1μ. At this level of magnification the image of the spores was clearly resolved. The spore concentration was adjusted beforehand so that the spores were fairly widely separated from each other. The movement of the slide was manipulated by a mechanical stage. To avoid biased selection of spores all the spores were measured which appeared completely within the field in the movement of the slide from left to right. Several samples of uredospores of each race were studied and one hundred spores were measured per sample.

Differences between the spores of different races were never significantly greater than differences between samples of the same race. Results obtained with one sample of spores are given in Table 13.

TABLE 13.

Uredospore Morphology (in μ) of Three Races Cultured under Identical Conditions and Collected 23rd May, 1953.

(Measured 23rd May, 1953.)

Race.	Average Length.	Range.	Average Breadth.	Range.
1	21·4	(18–25)	18·5	(15–21)
2	20·7	(16–24)	18·0	(15–20)
7	20·6	(16–25)	17·8	(15–20)

DISCUSSION.

The most striking feature of the race survey was the dichotomy of the Australian *Melampsora lini* race complex into the Punjab-attacking (PA) and non-Punjab-attacking (NPA) groups. This division paralleled differences in temperature tolerance in the field, and in vitro in the uredospore stage (Kerr, 1958), differences in host range in the field and in glasshouse tests, differences in the continuity of individual race members within each group in time and space.

None of the NPA races had so restricted a host range as PA race 1. Race 2, the least virulent of the NPA races, was highly virulent on four of the twenty differential

varieties. All the other NPA races were highly virulent on at least seven of the differential varieties. PA race 1 attacked only two of these varieties. J.W.S. gave an immune or fully susceptible reaction to the NPA races. Race 1 gave a variable resistant reaction even more marked in hybrids involving this variety. Bison was completely susceptible to all NPA races, as well as all races in America and New Zealand. It was immune to race 1.

The division between the PA and NPA groups was not an arbitrary classificatory division such as existed between the Ottawa-attacking and non-Ottawa-attacking races of Western Australia, determined by a single pathogenic factor. It seemed to be an important natural division. This might suggest that the difference was physiologic and that incompatibility prevented intergroup hybridization. This was ruled out by Charles's report (personal communication) of hybridization between races of the two groups in the glasshouse at Sydney University in 1947. There was no appreciable difference in uredospore morphology to indicate subspecific difference.

Hybridization between the two groups with progressive breakdown of the present differences might therefore have been expected. But there was no evidence that this had occurred since the first survey in 1940. Hybridization should eventually have produced among a wide range of races, races virulent on both Bison and Punjab, and others avirulent on both varieties. Since single factors condition virulence on these two varieties, the chances against the appearance of such races following hybridization were by no means great. Other widely virulent races, avirulent on Bison, and Punjab-attacking races lacking the high temperature tolerance of PA race 1 should also have appeared. The absence of such races from the field between 1940 and 1952 confirms the view that the PA and NPA race groups exist as two non-interpenetrating closed systems.

The existence of the two-group system poses several questions. How did the two systems originate? How is the dichotomy maintained and how long is it likely to continue? The answer to the second query is likely to suggest a solution to the first. If the difference did not stem from physiologic incompatibility it must have been maintained by factors external to the pathogen. Host and non-host environmental factors must have maintained the balance between the two groups.

The two groups did not occur together in the field on the same host varieties. The NPA group parasitized the commercial flax crops and were isolated on a number of occasions from the commercial linseed variety Walsh. The PA group, with one exception, was isolated from *Linum marginale*, a wild host, or such linseed varieties as Punjab and Imperial, which were not grown commercially and which were immune to all the NPA races. One collection of PA race 1 was received from Concurrent, a common host to the NPA group, but the plant yielding the race may well have been a rogue. This variety, fully susceptible to most NPA races from the seedling to the mature plant stage, was susceptible to PA race 1 in the seedling stage only. The difference in host range may have militated against hybridization of the two race groups.

The geographic distribution of the NPA group would seem to have been regulated primarily by temperature and to a lesser extent by host. There was an abundance of susceptible host in the southern flax-growing States. In the north the only commercial host was the linseed variety Walsh. Since this variety was generally resistant to the NPA races this might have militated against the epidemic development of the common NPA races in New South Wales and Queensland. But since the variety carried an appreciable percentage of off-type susceptible plants this could not account for the total absence of the NPA group from these States. Mid-summer temperatures in the linseed growing districts of these States exceeded the optimal, and possibly the maximum for development of the pathogen in the uredosoral state in the field. The combination of general host resistance and adverse summer temperatures probably accounted in large measure for the absence of the NPA groups. Summer temperatures in much of Victoria, South Australia, and Western Australia exceeded the optimal and possibly the maximum limits within which the NPA group could maintain itself

vegetatively in the uredosoral stage. But the more southern sections where flax was most commonly grown had a sufficiently mild summer to permit the group to aestivate successfully if not prolifically in this stage.

The occurrence of the PA group in all the flax- and linseed-growing parts of Australia, emphasized particularly by earlier race surveys, and also in districts where the commercial host was not grown or was restricted to very small experimental sowings presented one of the most interesting problems raised by the survey. Race 1, the only representative of this group identified during the current survey, was by far the most common race. It was the only Australian race found in all five States included in the survey. But it had the most restricted host range of all races identified during the survey. All commercial flax and linseed varieties were immune to it in the field. Such race 1-susceptible varieties as Punjab and Imperial were grown on far too small a scale and in too few localities for them to be considered the natural host of the PA group in the field. This suggested that the PA group did not maintain itself on the cultivated species, but rather on a widely distributed wild species. Such a host could be *Linum marginale*, native Australian flax. With the possible exception of parts of Queensland at a distance from the New South Wales border, this species was found in all flax- and linseed-growing districts of Australia, in many cooler highland districts bordering these districts, and at Tibooburra in inland New South Wales.

It would be interesting to know to what extent the distribution of the PA group was limited by higher summer temperatures. The common occurrence of the group in New South Wales and Queensland, at such places as Tibooburra in the former State and Biloela in the latter State, showed clearly that race 1 was much more tolerant to high summer temperatures in the field than NPA races. It was able to aestivate vegetatively in the uredosoral stage at Sydney, although an NPA race was completely eliminated from the field during the summer. Similar observations were made at Werribee in Victoria. In the linseed-growing zones of north-west New South Wales and Queensland, where summer temperatures are considerably higher, the pathogen may retreat to the cooler highland districts, just as the pathogen retreats from the hot plains of India (Prasada). While the only positive evidence from Queensland showed that race 1 did not survive in the uredosoral stage at Toowoomba beyond mid-summer, the race may well survive in cooler microlocal zones within such upland districts. The possibility of its survival on the inland plains was by no means ruled out, in view of the presence of the race at Tibooburra.

The mode of survival of the two race groups from year to year seemed to differ considerably. Within the NPA group there was little evidence that a given race maintained itself for long periods or spread to any great extent from its centre of origin. In view of the multiplicity of races during the current survey, and the marked change since earlier surveys, the sexual cycle must constitute a major phase in the history of this group. It seems unwise to assume that two collections of rust from different States or from the same State in different years were genetically identical and vegetatively related, even though they gave the same reaction on a given set of differential varieties.

Exhaustive race surveys were carried out by Cruickshank (1952, 1956) in New Zealand over what, by comparison with Australian conditions, was a relatively restricted area. There was a considerable carry-over of races in successive years. Since genetical investigations of host resistance and studies of uredospore longevity were being carried on simultaneously with the race survey, and since the writer was dependent on the kindness of field workers from other States for the supply of infected material, the same detailed survey of flax- and linseed-growing districts could not be carried out to assess the degree of carry-over of races within the NPA complex from one year to another. But a comparison of results obtained in the survey under discussion with results obtained in previous surveys, particularly in Western Australia, showed clearly that there had been a considerable shift in the NPA complex within a period of ten to thirteen years.

Ottawa-attacking races were restricted to a small section of Western Australia between 1949 and 1951. The first race, race 7, was identified in 1949, Another race, race 14, was found in 1950, and three new races, 15, 16 and 17, were isolated in 1951. One would judge from this evidence alone that the NPA complex was in a state of constant pathogenic flux. Race 14 was the first such race to be identified in another State, when it appeared in Victoria in 1952. It is impossible to determine whether the two occurrences of this race so separated in time and space stemmed from a common source or whether the race in Victoria developed *in situ*. But unless infected material was transferred between the two States it seems wisest to postulate independent origin.

Although a given race within the NPA complex may have failed to survive from one year to the next, new virulence factors, especially those with a high positive survival value, were not eliminated. The new factor conditioning virulence on Ottawa 770B soon became well established in the NPA complex of Western Australia even though the original race which carried it did not seem to have survived or spread. A factor or factors conditioning wide virulence on the heterogeneous variety Walsh appeared in race 11 in Victoria in 1949. The race did not seem to survive. Had it done so, specimens of such a potentially serious race would certainly have been forwarded from the district for analysis. But the new virulence factors evidently survived, though masked in less virulent races, eventually recombining in an important Walsh-attacking race reported from the same district in 1954.

The evident importance of the sexual cycle in the propagation of the NPA complex indicates the need for a careful study of this phase in the field. While there would not appear to be a Statewide suppression of the uredosoral stage in Victoria during summer, there did appear to be complete recession of this stage in some districts. This should increase the importance of the teleutospore stage as a carry-over phase. All stages in the life cycle of *M. lini* have been recorded on the cultivated host in New Zealand, as well as on the indigenous wild species *L. monogynum*. An intensive survey of southern flax-growing districts in Australia should indicate a like situation. This situation is most likely to obtain in the vicinity of flax mills, where harvested flax is stacked prior to processing. These stacks, by bringing together infected host and possibly different races from several districts, by buffering the teleutospore material against excesses of high summer temperature and ensuring a constant supply of volunteer plants, must provide those conditions by which the pathogenic potential of the pathogen is brought together, recombined and carried over from season to season. A visit to several flax mills near Melbourne in 1947 indicated an abundant supply of heavily infected volunteer plants.

It would be interesting to know to what extent the NPA race complex was affected by the growth cycle of the cultivated host. Mild temperatures and abundant supply of immature susceptible host ensured a maximum proliferation of this group during spring and early summer in Australia. High summer temperatures and lack of host following harvest resulted in an annual recession of the uredosoral stage, complete in some districts, partial in others according to local temperatures and supply of susceptible volunteer plants. Even if the recession were only partial and the volunteer plant population were entirely non-selective in its carry-over, it is highly improbable that the old balance of races would survive to the following year. The more severe the recession of the uredosoral stage in any year, the more prominent should be the role of the teleutospore as the carry-over phase, the more marked should be the change in the NPA complex in the following year. The diversity of races in the NPA complex and the marked change since the first survey could not, however, be attributed primarily to the Australian crop cycle. In New Zealand, with its much milder summer temperatures and spring sowings, *M. lini* maintained itself throughout the year in the vegetative uredosoral stage. The same multiplicity of races was found, though there was a considerable carry-over of old races in successive years.

The history of the PA group differed strikingly from the NPA group. Only one PA race, race 1, was detected during the four years of this survey. Specimens of this race were received from Western Australia, South Australia, Victoria, New South

Wales and Queensland. In the last two States it was located at such remote districts as Tibooburra and Biloela. Three Punjab-attacking races were identified in earlier surveys. They were differentiated by reactions on varieties not considered reliable for race differentiation in the current survey. Such differences as then existed were extremely fine and none of the three PA races had a range of virulence exceeding the least virulent of the NPA races. Such differences within the PA group as existed between 1940 and 1942 presumably still existed between 1948 and 1952, but could no longer be detected with the then used differential series and the currently recognized reaction levels of these varieties. Allowing for possible minor fluctuations of the nature indicated above there was no change in the pathogenicity of the PA complex from the first survey in 1940 to the completion of this survey in 1952. This contrasted with the NPA group within which fourteen of the eighteen races identified during this survey were recognizably different from races identified in earlier surveys. This suggested that the PA race group maintained itself entirely vegetatively in all parts of Australia or that the group, if it completed the sexual cycle, was pathogenically homozygous and homogeneous for those factors conditioning its avirulence on the eighteen immune members of the differential series. Both factors probably contributed to the uniformity of the PA group in time and space. Owing to the pressure of the several lines of investigation, race 1 teleutospores were not studied to determine the degree of homozygosity of this race. This could be a very useful study, which might throw much light on the unusual geographic and time patterning of this group.

Paralleling the remarkable pathogenic uniformity of the PA group was an equally notable uniformity in high temperature tolerance. This feature was lacking from the NPA group (Kerr, 1958) and must have contributed substantially to the PA group's capacity to penetrate and maintain itself in New South Wales and Queensland in a northerly sweep into higher temperature zones far beyond the line of the NPA group. If this tolerance were conditioned by a number of factors operating in complement, and some or all heterozygous, it might have contributed to the stability of the PA group by militating against the survival of off types lacking this tolerance. This seems unlikely since such races could have survived in the more temperate zones of the southern States. The mode of inheritance of high temperature tolerance could perhaps be determined relatively simply by studying the germination of uredospores of different segregates on host extract at temperatures in the vicinity of 20°C. (Kerr, 1958). It seems possible, assuming that the PA group completes the sexual cycle, that it is homozygous for those factors determining high-temperature tolerance, as well as those factors conditioning avirulence on the eighteen immune differential varieties. It would follow that mutations which might have resulted in the appearance of recessive factors conditioning virulence on some at least of these immune varieties have occurred very infrequently, if at all. With so many pathogenic factors involved it is surprising that no change in the pathogenicity of the PA group was registered over a period of twelve years.

Whether or not the sexual cycle played a major part in the survival of the PA group, it was evident that race 1 survived summer quite readily in the field in the uredosoral stage in areas where the NPA complex was suppressed. Given a year-round supply of susceptible host, the PA group could maintain itself vegetatively without difficulty in the southern States and in New South Wales, at least as far north as Sydney. This should certainly have tended to stabilize the pathogenicity of the group and might account in large measure for the uniformity of the group in time and space.

The manner of origin of new races is posed again by these studies, but no conclusive evidence can be brought forward to support either of the two major theories. In brief, these theories may be summarized as follows: (1) Mutations at loci conditioning virulence are relatively frequent. Potential new races, many with widened host range, are constantly being generated. (2) Mutations occur very infrequently. Any appreciable variation in virulence is the product of centuries of accumulation of mutant genes. Genes previously masked by dominant avirulent allels or present in too low a frequency to ensure detection, even in the most thorough survey, may

suddenly manifest themselves long after their origin when a change in the host complex suddenly confers a high positive survival value on races homozygous for such virulence factors.

Although no conclusive evidence for either theory can be drawn from the data presented in this paper, a line of investigations may be suggested which could lead to some definite decisions. The potential of such studies would depend to some extent on the validity of conclusions concerning the origin of the PA group. But even if such conclusions, to be dealt with shortly, are not valid, the study could yield useful information so long as the NPA and PA groups continue to coexist as non-interpenetrating systems.

Delaying consideration of this for the present it should be noted that while the evidence from the PA group tends to suggest that mutations are very infrequent, the evidence from the NPA group could be interpreted in favour of the theory that new races are constantly being generated by mutation of old races. Any evidence for this must assume that the change cannot be accounted for by introduction of new factors from outside sources. This assumes rigid policing of quarantine laws. The need for rigid quarantine measures has been fully recognized by those in a position to import seed. Such quarantine measures have become increasingly strict during the period 1940 to 1952, as flax and linseed assumed increasing economic importance. Prior to the war, when acreages were extremely low, lesser precautions seem to have been taken, and one outbreak at least of the pathogen in this period was attributed to contaminated imported seed.

While no natural quarantine barrier exists between the eastern States or these States and South Australia, there is an extensive desert barrier between Western Australia and the more easterly States. For this reason it seems reasonable to assume that the occurrence of an Ottawa-attacking race for the first time in Victoria several years after the first appearance of such a race in Western Australia represents an independent development of this race group. The development of this race group followed the release of new, supposedly resistant varieties, carrying Ottawa resistance. These varieties were developed within Australia. Had the appearance of the Ottawa-attacking group coincided with large-scale importation of seed this might have indicated that the race group traced its origin to the country of origin of this seed. That this was not so lends weight to the assumption that the group developed *in situ* in Western Australia. That Ottawa-attacking races probably developed independently in two States separated by a major quarantine barrier suggests that the Ottawa virulence factor either took its source in both States from recent mutations or was already present in the race complex in sufficient frequency to ensure its manifestation within a very short time of a change in the host from an unfavourable to a favourable population.

Since 1947 races avirulent on Abyssinian (races 2 and 4), avirulent on Williston Golden (race 17), and avirulent on Very Pale Blue Crimped (races 12 and 18) appeared in the Australian NPA complex for the first time. Previous evidence rules out the possibility that factors conditioning avirulence on these varieties were derived from the PA group. Genetic studies suggested that the Williston Golden factor conditioning resistance to PA race 1 differed from the factor conditioning resistance to NPA race 17. The new avirulence factors may have been introduced from overseas, though the introduction of three such factors in so short a time seems unlikely. They may have been present in the original race complex at a very low frequency and escaped detection. But since three factors were involved, since they could not have been masked by dominant allels, and since several hundred collections were analysed during earlier surveys this is by no means certain. The possibility of their origin by recent mutation cannot be ruled out.

It remains to determine the manner of origin of the two race groups. Many of the races now present in Australia stemmed from introduced races. McAlpine, reporting the first record of rust in this country, concluded that *M. lini* had been introduced from overseas. A rust epidemic in Victoria was attributed to the same cause in 1936.

Flor, in a personal communication, noted that the NPA races resembled those obtained by him by hybridization of North American and South American races. He suggested that races had been introduced from North America and Europe and had hybridized in Australia with more widely virulent races from South America.

The PA group almost certainly derived from some other source. Up to 1952 no such race as race 1, avirulent on Bison, had been recorded in North or South America. The avirulence of the North and South American races on Bolley Golden and Newland was conditioned by two factors and one factor respectively. The avirulence of PA race 1 on these varieties was conditioned by four and two factors respectively. Punjab-attacking races were identified in New Zealand, but none was avirulent on Bison (Cruickshank, personal communication). New Zealand Punjab-attacking races 3 and 13 (reclassified 10 and 8 respectively) (Cruickshank, 1956) were compared at Sydney with several Australian accessions of race 1. The New Zealand races were much less virulent on Punjab than the Australian accessions. *Linum marginale* was not included as a source of rust during the New Zealand race surveys (Cruickshank, personal communication), although it is an important hold-over host for the pathogen in that country. There is no information about the race complex present on this species, but the absence of Australian Punjab-attacking race types from commercial crops growing in the same districts as the wild species is in line with the assumption that the Australian PA group does not occur in New Zealand.

Indian races closely resembled the Australian PA group in their virulence on Bombay (carrying the same gene for resistance as Punjab) and avirulence on Bison and most of the differential varieties. If the Indian races possess the high temperature tolerance of the Australian PA group, and carry four factors and two factors respectively conditioning avirulence on Bolley Golden and Newland, the case for the derivation of the Australian PA group from India is strong.

This, however, cannot rule out the possibility that the PA group is endemic to Australia. Had the PA group been introduced in the same manner as the NPA group it is surprising that the two groups did not merge their pathogenic potential. Probably accounting for this and supplying the most critical evidence was the almost exclusive association of the native Australian flax *Linum marginale* with the PA group. Twelve of thirteen races collected from the wild species between 1940 and 1952 were Punjab-attacking races.

Wild flax species are common hosts to *M. lini* in all countries in which the pathogen has been studied, *L. monogynum* in New Zealand, *L. rigidum*, *L. lewisii*, *L. angustifolium* and *L. sulcatum* in America, as well as several species in Europe. It would be surprising if the native Australian species had only been host to *M. lini* since the settlement of the country. According to McAlpine, the pathogen was first identified on the cultivated host in 1889. Waterhouse obtained records of the pathogen on *L. marginale* at Como near Sydney in 1887. It seems reasonable to assume that the association of *M. lini* with the wild species predated the introduction of races from overseas. If this were so it is strange that the original race complex should be so repressed by introduced races that twelve of thirteen races isolated from the wild host between 1940 and 1952 were a race or races of Indian origin. The races isolated from the wild host may well have represented the original Australian *M. lini* complex. This was supported by the occurrence of race 1 on *L. marginale* at Tibooburra in the extreme north-west of New South Wales, a location so far removed from districts where the cultivated host was grown that derivation of the pathogen from the cultivated host seemed most unlikely in this instance. Identification of the pathogen at Pucuwan near Temora and at Curlewis added confirmation. Although Curlewis fell within areas where linseed was being grown commercially there was no known source of *M. lini* in the commercial crops. An exhaustive personal survey in the north-west of New South Wales, from Tamworth to Moree, failed to detect the slightest trace of rust in any of the commercial crops. Close checks by officers of the Department of Agriculture were equally fruitless. No commercial crops of flax or linseed were grown within 150 miles of Sydney, but a competent observer reported

that the wild species was commonly infected with rust in the Sydney area. The presence of the pathogen in New South Wales was almost certainly independent of the cultivated host.

The only evidence against these conclusions was the similarity of the Australian PA group and Indian races. This cannot be ignored, but it is by no means conclusive. It is not unreasonable to suppose that Australian and Indian races should be pathogenically similar. They might well trace back to a common source.

The association of the NPA complex with the cultivated host, *L. usitatissimum*, was reflected in the accumulation of a wide range of virulence factors specific for resistance factors carried by the various varieties within the species. The almost total lack of these factors in the PA group (stressed even more by the genetic investigations of host resistance to race 1) could be postulated as further evidence that the PA group was adapted to the wild species rather than the cultivated species. The PA group, despite its apparent pathogenic homogeneity, may well have accumulated a range of as yet undetected virulence factors specific for resistance factors in *L. marginale*. Since some lines of *marginale* were resistant to race 1 there must be diversity within the species. Should a thorough study of the wild species and the races present on it confirm the above, the case for an original Punjab-attacking *Melampsora lini* complex would be strong. No detailed study of the wild species has yet been made in Australia. Various lines were received from different States during these studies. Attempts were made to hybridize several of these lines with different varieties of *L. usitatissimum* using both species as female parents. The two species were completely incompatible.

If the wild species is the natural host of the PA group, this group should be able to maintain its distinctive identity as effectively in the future as it did between 1940 and 1952. A thorough study of this race complex might then indicate the extent to which the pathogen may generate new mutant virulence factors. This is impossible with the NPA complex where the possibility of introduction of new races from overseas can rarely be ruled out with complete certainty, and where the frequency of the individual virulence factors is controlled by not infrequent changes in the host complex. The presence of pathogenic factors in the PA complex specific for factors in the cultivated host could be used to determine the rate at which such factors are generated. Such factors, unless also fortuitously specific for or associated with some factor specific for resistance factors in the wild species, should be present at a frequency determined by the balance between the natural rates of generation and loss.

These studies confirm the well-established fact that a change in the host complex is generally followed by a change in the pathogen race complex. New varieties were released to counter the susceptibility of the common flax varieties Concurrent and Liral Crown. Shortly after the release of Wada and Boyup, Ottawa-attacking races appeared for the first time in Australia and soon dominated the Western Australia NPA complex. This stresses the need for combined resistance already emphasized by Watson and Singh (1952).

The range of combined resistance in *Linum usitatissimum* is greatly reduced by the limited number of loci in the host determining resistance to *Melampsora lini*. But this may be offset to some extent by the constant pathogenic flux of the NPA group. Flor (1946) established a one-to-one correlation between resistance factors in the host and specific virulence factors in the pathogen. Avirulence was always dominant to virulence. The greater the number of resistance factors in a host variety, the less likely is the pathogen to accumulate the requisite virulence factors homozygously in the one race. If the pathogen survives via the teleutospore stage rather than vegetatively from one season to another, the survival of a given complex of virulence factors becomes increasingly uncertain in a mixed race situation the greater the number of virulence factors involved.

During these studies, Wada, with its resistance derived from a single Ottawa factor, succumbed to an Ottawa-attacking race soon after its release for commercial production. While the original race did not survive, the Ottawa-attacking virulence

factor carried over in the race complex from one year to the next in Western Australia. Walsh, by contrast, the only commercially grown linseed variety, grown for a considerable period over a wide geographic range, remained effectively immune or resistant to the Australian PA and NPA race groups. One Walsh-attacking race appeared in one district in Victoria, but did not spread or survive to the next year. Genetic studies have shown that Walsh, though heterozygous, owes its resistance to at least three factors.

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EXPLANATION OF PLATES III–IV.

Plate iii.

1.—Fully susceptible type 4 reaction. Compound pustules with no chlorosis. Leaves rarely distorted. Note small newly developing non-compound pustules on same leaf. ×3½.

2.—Typical fully susceptible reaction without compound pustules. No chlorosis and infection general over whole leaf surface. ×3½.

3.—Fleck type immune reaction. Small chlorotic flecks over most of leaf surface. × 3½.

4, 5, 6.—Various intermediate resistant reactions with pustules of varying size and varying degrees of chlorosis and necrosis. Infection often localized to part of the leaf, and leaf commonly misshapen. × 3½.

Plate iv.

1.—Bulk inoculation of F257 with race 1. Fairly heavy production of uredosori on leaves, but very few developing on stems. Plants recovered and still growing vigorously. × ⅔.

2.—Two sets of excised shoots of Concurrent inoculated and incubated at the same time under identical conditions with an Ottawa-attacking race, race 17 (LHS), and a non-Ottawa-attacking race, race 6 (RHS), Note complete recovery of shoots inoculated with race 17 and the clean uninfected stems. Shoots inoculated with race 6 developed heavy stem infection and failed to resume normal growth. × ⅓.

3.—Bulk inoculation of Punjab with race 1. Very heavy development of uredosori on leaves and stem. Growth of shoots severely retarded. × ⅔.